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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
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NEWS	5	NOV 30	PHAR reloaded with additional data
NEWS	6	DEC 01	LISA now available on STN
NEWS	7	DEC 09	12 databases to be removed from STN on December 31, 2004
NEWS	8	DEC 15	MEDLINE update schedule for December 2004
NEWS	9	DEC 17	ELCOM reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	10	DEC 17	COMPUAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	11	DEC 17	SOLIDSTATE reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	12	DEC 17	CERAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	13	DEC 17	THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
NEWS	14	DEC 30	EPFULL: New patent full text database to be available on STN
NEWS	15	DEC 30	CAPLUS - PATENT COVERAGE EXPANDED
NEWS	16	JAN 03	No connect-hour charges in EPFULL during January and February 2005
NEWS	17	JAN 11	CA/CAPLUS - Expanded patent coverage to include Russia (Federal Institute of Industrial Property)

NEWS EXPRESS	JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005
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FILE 'HOME' ENTERED AT 14:40:58 ON 21 JAN 2005

=> FIL MEDLINE BIOSIS EMBASE CA SCISEARCH
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 14:41:06 ON 21 JAN 2005

FILE 'BIOSIS' ENTERED AT 14:41:06 ON 21 JAN 2005
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=> s cd63
L1         3072 CD63

=> s l1 and macrophag?
L2          269 L1 AND MACROPHAG?

=> s l2 and (antibod? or anti-bod? or antiser? or anti-ser?)
   3 FILES SEARCHED...
L3          113 L2 AND (ANTIBOD? OR ANTI-BOD? OR ANTISER? OR ANTI-SER?)

=> s l3 and (macrophag? (5n) ex vivo)
L4            0 L3 AND (MACROPHAG? (5N) EX VIVO)

=> s l1 and ((ANTIBOD? OR ANTI-BOD? OR ANTISER? OR ANTI-SER?) (5n) ex vivo)
   3 FILES SEARCHED...
L5            0 L1 AND ((ANTIBOD? OR ANTI-BOD? OR ANTISER? OR ANTI-SER?) (5N)
                EX VIVO)

=> s (ANTIBOD? OR ANTI-BOD? OR ANTISER? OR ANTI-SER?)
   3 FILES SEARCHED...
L6       2804388 (ANTIBOD? OR ANTI-BOD? OR ANTISER? OR ANTI-SER?)

=> s l1 (5n) l6
L7          222 L1 (5N) L6

=> s l7 and (ex vivo)
L8            6 L7 AND (EX VIVO)

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9            2 DUP REM L8 (4 DUPLICATES REMOVED)

=> s l9 and py=<2000
   2 FILES SEARCHED...
L10           1 L9 AND PY=<2000

=> d l10 ibib abs

L10  ANSWER 1 OF 1  BIOSIS  COPYRIGHT (c) 2005 The Thomson Corporation.  on STN
ACCESSION NUMBER:    1999:262237  BIOSIS
DOCUMENT NUMBER:     PREV199900262237
```

TITLE: 1-Deamino (8-D-arginine) vasopressin infusion partially corrects platelet deposition on subendothelium in Bernard-Soulier syndrome: The role of factor VIII.
AUTHOR(S): Lozano, M. [Reprint author]; Escolar, G.; Bellucci, S.; Monteagudo, J.; Pico, M.; Ordinas, A.; Caen, J. P.
CORPORATE SOURCE: Department of Hemotherapy and Hemostasis, Hospital Clinic, Villarroel 170, 08036, Barcelona, Spain
SOURCE: Platelets (Abingdon), (1999) Vol. 10, No. 2-3, pp. 141-145. print.
ISSN: 0953-7104.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Jul 1999
Last Updated on STN: 15 Jul 1999

AB The mechanism of the transient beneficial effect of 1-deamino(8-D-arginine) vasopressin (dDAVP) infusion in the hemostasis of some BSS patients is not fully understood. We have studied the effect of dDAVP infusion in a BSS patient using an **ex vivo** perfusion system. Additional coagulation and flow cytometry studies were also performed. Prolonged bleeding time (> 30 min) was not affected by dDAVP infusion. However, perfusion experiments performed with low molecular weight heparin anticoagulated blood (which permits the study of fibrin deposition on perfused subendothelium) showed a significant increase in platelet deposition (6.2% before dDAVP infusion; 20.3% after) and fibrin formation. dDAVP infusion also caused an increase in prothrombin consumption compared with base line values (33 vs 46%). Flow cytometry studies of the patients platelets showed no changes in binding of monoclonal **antibodies** against CD41, CD36, CD62P or **CD63** . The increase in thrombus formation observed in perfusions may be dependent on FVIII since it could be reproduced by adding purified free or von Willebrand factor (vWf)-associated FVIII to the patient's blood in vitro. The shortening effect of dDAVP on bleeding time observed in some Bernard-Soulier syndrome patients might be related to an increase in factor FVIII levels induced by dDAVP infusion.

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=> d his

     (FILE 'HOME' ENTERED AT 14:40:58 ON 21 JAN 2005)

     FILE 'MEDLINE, BIOSIS, EMBASE, CA, SCISEARCH' ENTERED AT 14:41:06 ON 21
     JAN 2005
L1          3072 S CD63
L2           269 S L1 AND MACROPHAG?
L3           113 S L2 AND (ANTIBOD? OR ANTI-BOD? OR ANTISER? OR ANTI-SER?)
L4             0 S L3 AND (MACROPHAG? (5N) EX VIVO)
L5             0 S L1 AND ((ANTIBOD? OR ANTI-BOD? OR ANTISER? OR ANTI-SER?) (5N)
L6       2804388 S (ANTIBOD? OR ANTI-BOD? OR ANTISER? OR ANTI-SER?)
L7           222 S L1 (5N) L6
L8             6 S L7 AND (EX VIVO)
L9             2 DUP REM L8 (4 DUPLICATES REMOVED)
L10            1 S L9 AND PY=<2000

=> s l7 (5n) ((in vivo) or (whole animal?))
   2 FILES SEARCHED...
L11            0 L7 (5N) ((IN VIVO) OR (WHOLE ANIMAL?))

=> s l7 (s) ((in vivo) or (whole animal?) or rat or mouse or mice)
   2 FILES SEARCHED...
L12           21 L7 (S) ((IN VIVO) OR (WHOLE ANIMAL?) OR RAT OR MOUSE OR MICE)

=> dup rem l12; s l12 not l9
PROCESSING COMPLETED FOR L12
L13            6 DUP REM L12 (15 DUPLICATES REMOVED)
```

L14 21 L12 NOT L9

=> s l13 not l9
L15 6 L13 NOT L9

=> d l15 ibib abs 1-6

L15 ANSWER 1 OF 6 MEDLINE on STN
ACCESSION NUMBER: 2000136113 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10669631
TITLE: CD9 participates in endothelial cell migration during in vitro wound repair.
AUTHOR: Klein-Soyer C; Azorsa D O; Cazenave J P; Lanza F
CORPORATE SOURCE: INSERM U. 311, Etablissement de Transfusion Sanguine de Strasbourg Strasbourg, France.. claudine.soyer@etss.u-strasbg.fr
SOURCE: Arteriosclerosis, thrombosis, and vascular biology, (2000 Feb) 20 (2) 360-9.
Journal code: 9505803. ISSN: 1079-5642.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000320
Last Updated on STN: 20000320
Entered Medline: 20000303

AB CD9, a widely expressed membrane protein of the tetraspanin family, has been implicated in diverse functions, such as signal transduction, cell adhesion, and cell motility. We tested the effects of an anti-CD9 monoclonal antibody (ALMA.1) on the migration and proliferation of human vascular endothelial cells (ECs) during repair of an in vitro mechanical wound mimicking angiogenic processes. ALMA.1 induced dose-dependent inhibition of wound repair with a 35+/-1.5% decrease at 20 microg/mL. Only cell migration was affected, because the rate of proliferation of ECs at the lesion margin was not modified and because the inhibition of repair was also observed for nonproliferating irradiated ECs. Monoclonal **antibodies** against **CD63** tetraspanin (H5C6) and control **mouse** IgG (MOPC-21) were inactive. CD9, one of the most abundant proteins at the surface of ECs, colocalized with beta(1) or beta(3) integrins on EC membranes in double-labeling immunofluorescence experiments with ALMA.1 and an anti-beta(1) (4B4) or anti-beta(3) (SDF.3) monoclonal antibody. Moreover, ALMA.1 and 4B4 had additive inhibitory effects on lesion repair, whereas 4B4 alone also inhibited EC proliferation. In transmembrane Boyden-type assays, ALMA.1 induced dose-dependent inhibition of EC migration toward fibronectin and vitronectin with 45+/-6% and 31+/-10% inhibition, respectively, at 100 microg/mL. 4B4 inhibited migration toward fibronectin at 10 microg/mL but had no effect in the case of vitronectin. Adhesion of ECs to immobilized anti-CD9 monoclonal antibodies induced tyrosine-phosphorylated protein levels similar to those observed during interactions with beta(1) or beta(3) integrins. These results point to the involvement of CD9 in EC adhesion and migration during lesion repair and angiogenesis, probably through cooperation with integrins. As such, CD9 is a potential target to inhibit angiogenesis in metastatic and atherosclerotic processes.

L15 ANSWER 2 OF 6 MEDLINE on STN
ACCESSION NUMBER: 1999105712 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9890706
TITLE: Monoclonal **antibody** to **rat CD63** detects different molecular forms in **rat** tissue.
AUTHOR: Kennel S J; Lankford P K; Foote L J; Davis I A

CORPORATE SOURCE: Life Sciences Division, Oak Ridge National Laboratory, TN 37831-6101, USA.
SOURCE: Hybridoma, (1998 Dec) 17 (6) 509-15.
Journal code: 8202424. ISSN: 0272-457X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990326
Last Updated on STN: 19990326
Entered Medline: 19990316

AB From mice immunized with rat endothelial cell membranes, we isolated several hybridomas secreting monoclonal antibodies (MAbs) to a 45-kDa glycoprotein expressed on the surface of cultured cells. One of these antibodies, 523-14A, was purified and used for immunoaffinity chromatography, Western blotting, and immunohistochemistry. The glycoprotein containing the antigen for MAb 523-14A, gp45, was isolated from rat lung endothelial cell membranes using wheat germ agglutinin and antibody affinity chromatography sequentially. Mass spectrometry of tryptic peptides from gel purified bands identified gp45 as rat CD63, a member of the transmembrane-4 superfamily. Western blot analyses of tissues from F344 rats showed that kidney, spleen, uterus, and ovaries expressed CD63 at high levels. Thymus, salivary gland, testicles, intestines, pancreas, and adrenals expressed lower amounts. Tissue cell types expressing CD63 were also examined and the results showed that, in addition to the expected expression on lymphoid cells, CD63 was expressed on many epithelial and muscle cells as well. The mobility of CD63 on SDS-PAGE varied widely, indicative of molecular masses ranging from 45 kDa in some tissues to nearly 60 kDa in others.

L15 ANSWER 3 OF 6 MEDLINE on STN
ACCESSION NUMBER: 96256884 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8643103
TITLE: **Antibodies** against human **CD63** activate transfected **rat** basophilic leukemia (RBL-2H3) cells.
AUTHOR: Smith D A; Monk P N; Partridge L J
CORPORATE SOURCE: Krebs Institute for Biomolecular Research, Department of Molecular Biology and Biotechnology, University of Sheffield, UK.
SOURCE: Molecular immunology, (1995 Dec) 32 (17-18) 1339-44.
Journal code: 7905289. ISSN: 0161-5890.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199607
ENTRY DATE: Entered STN: 19960726
Last Updated on STN: 19970203
Entered Medline: 19960715

AB CD63 is a widely expressed glycoprotein member of the transmembrane 4 superfamily (TM4SF) that is present on activated platelets, monocytes and macrophages and many non-lymphoid cells. It has been proposed that CD63 and other members of the TM4SF couple to intracellular signal transduction pathways and may have a role in cellular adhesion, proliferation and activation. We have investigated the functions of human CD63 by expression in the **rat** basophilic leukemia cell line, RBL-2H3, which has previously been reported to respond to **antibodies** against the **rat** homolog of **CD63.** Using a panel of antibodies against human CD63 we have shown that high levels of granular secretion from transfected RBL cells can be stimulated by some, but not all, of the antibodies. The specificity of this response suggests that these activating antibodies may be mimicking a natural ligand for CD63.

The secretory response to crosslinking of the high affinity IgE receptor and also that to non-receptor stimuli (phorbol ester and calcium ionophore) is inhibited by an antibody that appears to recognise both human and rat homologs of CD63. These results suggest that stimulus-secretion coupling can occur through human CD63 and that RBL cells transfected with this protein will constitute a valuable tool in elucidating its function.

L15 ANSWER 4 OF 6 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2002188958 EMBASE
TITLE: An alternate targeting pathway for procathepsin L in mouse fibroblasts.
AUTHOR: Ahn K.; Yeyeodu S.; Collette J.; Madden V.; Arthur J.; Li L.; Erickson A.H.
CORPORATE SOURCE: A.H. Erickson, Department of Biochemistry, The University of North Carolina, Chapell Hill, NC 27599-7260, United States. erickson@unc.edu
SOURCE: Traffic, (2002) 3/2 (147-159).
Refs: 68
ISSN: 1398-9219 CODEN: TRAFFA
COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB In transformed **mouse** fibroblasts, a significant proportion of the lysosomal cysteine protease cathepsin L remains in cells as an inactive precursor which associates with membranes by a mannose phosphate-independent interaction. When microsomes prepared from these cells were resolved on sucrose gradients, this procathepsin L was localized in dense vesicles distinct from those enriched for growth hormone, which is secreted constitutively when expressed in fibroblasts. Ultrastructural studies using antibodies directed against the propeptide to avoid detection of the mature enzyme in lysosomes revealed that the proenzyme was concentrated in dense cores within small vesicles and multivesicular endosomes which labeled with **antibodies** specific for **CD63.** Consistent with the resemblance of these cores to those of regulated secretory granules, secretion of procathepsin L from fibroblasts was modestly stimulated by phorbol, 12-myristate, 13-acetate. When protein synthesis was blocked with cycloheximide and lysosomal proteolysis inhibited with leupeptin, procathepsin L was found to gradually convert to the active single-chain protease. The data suggest that when synthesis levels are high, a portion of the procathepsin L is packaged in dense cores within multivesicular endosomes localized near the plasma membrane. Gradual activation of this proenzyme achieves targeting of the proenzyme to lysosomes by a mannose phosphate receptor-independent pathway.

L15 ANSWER 5 OF 6 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 1999339897 EMBASE
TITLE: Y receptor-mediated induction of CD63 transcripts, a tetraspanin determined to be necessary for differentiation of the intestinal epithelial cell line, hBRIE 380i cells.
AUTHOR: Hallden G.; Hadi M.; Hong H.T.; Aponte G.W.
CORPORATE SOURCE: G.W. Aponte, Dept. of Nutritional Sciences, 119 Morgan Hall, University of California, Berkeley, CA 94720-3104, United States. gwa@nature.berkeley.edu
SOURCE: Journal of Biological Chemistry, (24 Sep 1999) 274/39 (27914-27924).
Refs: 89
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Peptide YY (PYY) and neuropeptide Y (NPY) are peptides that coordinate intestinal activities in response to luminal and neuronal signals. In this study, using the **rat** hybrid small intestinal epithelial cell line, hBRIE 380i cells, we demonstrated that PYY- and NPY-induced rearrangement of actin filaments may be in part through a Y1α and/or a nonneuronal Y2 receptor, which were cloned from both the intestinal mucosa and the hBRIE 380i cells. A number of PYY/NPY-responsive genes were also identified by subtractive hybridization of the hBRIE 380i cells in the presence or absence of a 6-h treatment with PYY. Several of these genes coded for proteins associated with the cell cytoskeleton or extracellular matrix. One of these proteins was the transmembrane-4 superfamily protein CD63, previously shown to associate with β1-integrin and implicated in cell adhesion. **CD63** immunoreactivity, using **antibody** to the extracellular domain, was highest in the differentiated cell clusters of the hBRIE 380i cells. The hBRIE 380i cells transfected with antisense CD63 cDNA lost these differentiated clusters. These studies suggest a new role for NPY and PYY in modulating differentiation through cytoskeletal associated proteins.

L15 ANSWER 6 OF 6 EMBASE
on STN

ACCESSION NUMBER: 1999138385 EMBASE
TITLE: L-Deamino (8-D-arginine) vasopressin infusion partially corrects platelet deposition on subendothelium in Bernard-Soulier syndrome: The role of factor VIII.
AUTHOR: Lozano M.; Escolar G.; Bellucci S.; Monteagudo J.; Pico M.; Ordinas A.; Caen J.P.
CORPORATE SOURCE: Dr. M. Lozano, Hospital Clinic, Department of Hemotherapy Hemostasis, Villarroel 170, 08036 Barcelona, Spain. mlozano@medicina.ub.es
SOURCE: Platelets, (1999) 10/2-3 (141-145).
Refs: 34
ISSN: 0953-7104 CODEN: PLTEEF
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 025 Hematology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The mechanism of the transient beneficial effect of 1-deamino(8-D-arginine) vasopressin (dDAVP) infusion in the hemostasis of some BSS patients is not fully understood. We have studied the effect of dDAVP infusion in a BSS patient using an ex **vivo** perfusion system. Additional coagulation and flow cytometry studies were also performed. Prolonged bleeding time (> 30 min) was not affected by dDAVP infusion. However, perfusion experiments performed with low molecular weight heparin anticoagulated blood (which permits the study of fibrin deposition on perfused subendothelium) showed a significant increase in platelet deposition (6.2% before dDAVP infusion; 20.3% after) and fibrin formation. dDAVP infusion also caused an increase in prothrombin consumption compared with base line values (33 vs 46%). Flow cytometry studies of the patients platelets showed no changes in binding of monoclonal **antibodies** against CD41, CD36, CD62P or **CD63.** The increase in thrombus formation observed in perfusions may be dependent on FVIII since it could be reproduced by adding purified free or von Willebrand factor (vWf)-associated FVIII to the patient's blood in vitro. The shortening effect of dDAVP on bleeding time observed in some Bernard-Soulier syndrome patients might be related to an increase in factor FVIII levels induced by dDAVP infusion.

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FULL ESTIMATED COST	114.19	114.40

STN INTERNATIONAL LOGOFF AT 15:02:09 ON 21 JAN 2005